

Posttraumatic stress disorder

Posttraumatic stress disorder (PTSD)^[note 1] is a mental disorder that can develop after a person is exposed to a traumatic event, such as sexual assault, warfare, traffic collisions, or other threats on a person's life.^[1] Symptoms may include disturbing thoughts, feelings, or dreams related to the events, mental or physical distress to trauma-related cues, attempts to avoid trauma-related cues, alterations in how a person thinks and feels, and an increase in the fight-or-flight response.^{[1][3]} These symptoms last for more than a month after the event.^[1] Young children are less likely to show distress but instead may express their memories through play.^[1] A person with PTSD is at a higher risk for suicide and intentional self-harm.^{[2][6]}

Most people who have experienced a traumatic event will not develop PTSD.^[2] People who experience interpersonal trauma (for example rape or child abuse) are more likely to develop PTSD, as compared to people who experience non-assault based trauma such as accidents and natural disasters.^[7] About half of people develop PTSD following rape.^[2] Children are less likely than adults to develop PTSD after trauma, especially if they are under ten years of age.^[8] Diagnosis is based on the presence of specific symptoms following a traumatic event.^[2]

Prevention may be possible when therapy is targeted at those with early symptoms but is not effective when carried out among all people following trauma.^[2] The main treatments for people with PTSD are counselling and medication.^[3] A number of different types of therapy may be useful.^[9] This may occur one-on-one or in a group.^[3] Antidepressants of the selective serotonin reuptake inhibitor type are the first-line medications for PTSD and result in benefit in about half of people.^[4] These benefits are less than those seen with therapy.^[2] It is unclear if using medications and therapy together has greater benefit.^{[2][10]} Other medications do not have enough evidence to support their use and in the case of benzodiazepines may worsen outcomes.^{[11][12]}

In the United States about 3.5% of adults have PTSD in a given year, and 9% of people develop it at some point in their life.^[1] In much of the rest of the world, rates during a given year are between 0.5% and 1%.^[1] Higher rates may occur in regions of armed conflict.^[2] It is more common in women than men.^[3]

Symptoms of trauma-related mental disorders have been documented since at least the time of the ancient Greeks.^[13] During the World Wars study increased and it was known under various terms including "shell shock" and "combat

Posttraumatic stress disorder



Art therapy project created by a U.S. Marine with posttraumatic stress disorder

Specialty	Psychiatry, clinical psychology
Symptoms	Disturbing thoughts, feelings, or dreams related to the event; mental or physical distress to trauma-related cues; efforts to avoid trauma-related situations; increased fight-or-flight response ^[1]
Complications	Suicide ^[2]
Duration	> 1 month ^[1]
Causes	Exposure to a traumatic event ^[1]
Diagnostic method	Based on symptoms ^[2]
Treatment	Counseling, medication ^[3]
Medication	Selective serotonin reuptake inhibitor ^[4]
Frequency	8.7% (lifetime risk); 3.5% (12-month risk) (USA) ^[5]

neurosis".^[14] The term "posttraumatic stress disorder" came into use in the 1970s in large part due to the diagnoses of U.S. military veterans of the Vietnam War.^[15] It was officially recognized by the American Psychiatric Association in 1980 in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III).^[16]

Contents

Signs and symptoms

Associated medical conditions

Risk factors

Trauma

Genetics

Pathophysiology

Neuroendocrinology

Neuroanatomy

Diagnosis

Screening and assessment

Diagnostic and statistical manual

International classification of diseases

Differential diagnosis

Prevention

Psychological debriefing

Risk-targeted interventions

Management

Psychotherapy

Medication

Other

Epidemiology

United States

Veterans

United States

United Kingdom

Canada

History

Terminology

Research

Psychotherapy adjuncts

Lawsuits

Notes

References

External links

Video explanation

Signs and symptoms

Symptoms of PTSD generally begin within the first 3 months after the inciting traumatic event, but may not begin until years later.^{[1][3]} In the typical case, the individual with PTSD persistently avoids trauma-related thoughts and emotions, and discussion of the traumatic event, and may even have amnesia of the event. However, the event is commonly relived by the individual through intrusive, recurrent recollections, flashbacks, and nightmares.^[17] While it is common to have symptoms after any traumatic event, these must persist to a sufficient degree (i.e., causing

dysfunction in life or clinical levels of distress) for longer than one month after the trauma to be classified as PTSD (clinically significant dysfunction or distress for less than one month after the trauma may be acute stress disorder).^{[1][18][19][20]}

Associated medical conditions

Drug abuse and alcohol abuse commonly co-occur with PTSD.^[21] Recovery from posttraumatic stress disorder or other anxiety disorders may be hindered, or the condition worsened, when substance use disorders are comorbid with PTSD. Resolving these problems can bring about improvement in an individual's mental health status and anxiety levels.^{[22][23]}

Risk factors

Persons considered at risk include combat military personnel, victims of natural disasters, concentration camp survivors, and victims of violent crime. Persons employed in occupations that expose them to violence (such as soldiers) or disasters (such as emergency service workers) are also at risk.^[25] Other occupations that are at higher risk include police officers, firefighters, ambulance personnel, health care professionals, train drivers, divers, journalists, and sailors, in addition to people who work at banks, post offices or in stores.^[26] The size of the hippocampus is inversely related to post-traumatic stress disorder and treatment success; the smaller the hippocampus, the higher risk of PTSD.^[27]

Trauma

PTSD has been associated with a wide range of traumatic events. The risk of developing PTSD after a traumatic event varies by trauma type^{[28][29]} and is highest following exposure to sexual violence (11.4%), particularly rape (19.0%).^[30] Men are more likely to experience a traumatic event, but women are more likely to experience the kind of high-impact traumatic event that can lead to PTSD, such as interpersonal violence and sexual assault.^[31]

Posttraumatic stress reactions have not been studied as well in children and adolescents as adults.^[8] The rate of PTSD may be lower in children than adults, but in the absence of therapy, symptoms may continue for decades.^[8] One estimate suggests that the proportion of children and adolescents having PTSD in a non-wartorn population in a developed country may be 1% compared to 1.5% to 3% of adults, and much lower below the age of 10 years.^[8] On average, 16% of children exposed to a traumatic event develop PTSD, varying according to type of exposure and gender.^[32]

Predictor models have consistently found that childhood trauma, chronic adversity, and familial stressors increase risk for PTSD as well as risk for biological markers of risk for PTSD after a traumatic event in adulthood.^{[33][34][35]} Experiencing bullying as a child or an adult has been correlated with the development of PTSD.^[36] Peritraumatic dissociation in children is a predictive indicator of the development of PTSD later in life.^[37] This effect of childhood trauma, which is not well understood, may be a marker for both traumatic experiences and attachment problems.^{[38][39]} Proximity to, duration of, and severity of the trauma make an impact, and interpersonal traumas cause more problems than impersonal ones.^[40]



Service members use art to relieve PTSD symptoms.



No quieren (*They do not want to*) by Francisco Goya (1746–1828) depicts an elderly woman wielding a knife in defense of a girl being assaulted by a soldier.^[24]

The risk of developing PTSD is increased in individuals who are exposed to physical abuse, physical assault, or kidnapping.^{[41][42]} Women who experience physical violence are more likely to develop PTSD than men.^[41]

Intimate partner violence

An individual that has been exposed to domestic violence is predisposed to the development of PTSD. However, being exposed to a traumatic experience does not automatically indicate that an individual will develop PTSD.^[43] There is a strong association between the development of PTSD in mothers that experienced domestic violence during the perinatal period of their pregnancy.^[44]

Those who have experienced sexual assault or rape may develop symptoms of PTSD.^{[45][46]} PTSD symptoms include re-experiencing the assault, avoiding things associated with the assault, numbness, and increased anxiety and an increased startle response. The likelihood of sustained symptoms of PTSD is higher if the rapist confined or restrained the person, if the person being raped believed the rapist would kill him or her, the person who was raped was very young or very old, and if the rapist was someone he or she knew. The likelihood of sustained severe symptoms is also higher if people around the survivor ignore (or are ignorant of) the rape or blame the rape survivor.^[47]

War-related trauma

Military service is a risk factor for developing PTSD.^[48] Around 78% of people exposed to combat do not develop PTSD; in about 25% of military personnel who develop PTSD, its appearance is delayed.^[48]

Refugees are also at an increased risk for PTSD due to their exposure to war, hardships, and traumatic events. The rates for PTSD within refugee populations range from 4% to 86%.^[49] While the stresses of war impact everyone involved, displaced persons have been shown to be more affected than nondisplaced persons.^[50]

Unexpected death of a loved one

Sudden, unexpected death of a loved one is the most common traumatic event type reported in cross-national studies.^{[30][51]} However, the majority of people who experience this type of event will not go on to develop PTSD. An analysis from the WHO World Mental Health Surveys found a 5.2% risk of developing PTSD after learning of the unexpected death of a loved one.^[51] Because of the high prevalence of this type of traumatic event, unexpected death of a loved one accounts for approximately 20% of PTSD cases worldwide.^[30]

Life-threatening illness

Medical conditions associated with an increased risk of PTSD include cancer,^{[52][53][54]} heart attack,^[55] and stroke.^[56] Intensive-care unit (ICU) hospitalization is also a risk factor for PTSD.^[57] Some women experience PTSD from their experiences related to breast cancer and mastectomy.^{[58][59][52]}

Pregnancy-related trauma

Women who experience miscarriage are at risk of PTSD.^{[60][61][62]} Those who experience subsequent miscarriages have an increased risk of PTSD compared to those experiencing only one.^[60] PTSD can also occur after childbirth and the risk increases if a woman has experienced trauma prior to the pregnancy.^{[63][64]} Prevalence of PTSD following normal childbirth (that is, excluding stillbirth or major complications) is estimated to be between 2.8 and 5.6% at 6 weeks



A U.S. Long-Range Patrol team leader in Vietnam, 1968.

postpartum,^[65] with rates dropping to 1.5% at 6 months postpartum.^{[65][66]} Symptoms of PTSD are common following childbirth, with prevalence of 24-30.1%^[67] at 6 weeks, dropping to 13.6% at 6 months.^[68] Emergency childbirth is also associated with PTSD.^[69] Some women experience PTSD from their experiences related to breast cancer and mastectomy.^{[58][70][52]}

Genetics

There is evidence that susceptibility to PTSD is hereditary. Approximately 30% of the variance in PTSD is caused from genetics alone.^[37] For twin pairs exposed to combat in Vietnam, having a monozygotic (identical) twin with PTSD was associated with an increased risk of the co-twin's having PTSD compared to twins that were dizygotic (non-identical twins).^[71] There is evidence that those with a genetically smaller hippocampus are more likely to develop PTSD following a traumatic event. Research has also found that PTSD shares many genetic influences common to other psychiatric disorders. Panic and generalized anxiety disorders and PTSD share 60% of the same genetic variance. Alcohol, nicotine, and drug dependence share greater than 40% genetic similarities.^[37]

Several biological indicators have been identified that are related to later PTSD development. Heightened startle responses and a smaller hippocampal volume have been identified as biomarkers for the risk of developing PTSD.^[27] Additionally, one study found that soldiers whose leukocytes had greater numbers of glucocorticoid receptors were more prone to developing PTSD after experiencing trauma.^[72]

Pathophysiology

Neuroendocrinology

PTSD symptoms may result when a traumatic event causes an over-reactive adrenaline response, which creates deep neurological patterns in the brain. These patterns can persist long after the event that triggered the fear, making an individual hyper-responsive to future fearful situations.^{[18][73]} During traumatic experiences the high levels of stress hormones secreted suppress hypothalamic activity that may be a major factor toward the development of PTSD.^[74]

PTSD causes biochemical changes in the brain and body, that differ from other psychiatric disorders such as major depression. Individuals diagnosed with PTSD respond more strongly to a dexamethasone suppression test than individuals diagnosed with clinical depression.^{[75][76]}

Most people with PTSD show a low secretion of cortisol and high secretion of catecholamines in urine,^[77] with a norepinephrine/cortisol ratio consequently higher than comparable non-diagnosed individuals.^[78] This is in contrast to the normative fight-or-flight response, in which both catecholamine and cortisol levels are elevated after exposure to a stressor.^[79]

Brain catecholamine levels are high,^[80] and corticotropin-releasing factor (CRF) concentrations are high.^{[81][82]} Together, these findings suggest abnormality in the hypothalamic-pituitary-adrenal (HPA) axis.

The maintenance of fear has been shown to include the HPA axis, the locus coeruleus-noradrenergic systems, and the connections between the limbic system and frontal cortex. The HPA axis that coordinates the hormonal response to stress,^[83] which activates the LC-noradrenergic system, is implicated in the over-consolidation of memories that occurs in the aftermath of trauma.^[84] This over-consolidation increases the likelihood of one's developing PTSD. The amygdala is responsible for threat detection and the conditioned and unconditioned fear responses that are carried out as a response to a threat.^[37]

The HPA axis is responsible for coordinating the hormonal response to stress.^[37] Given the strong cortisol suppression to dexamethasone in PTSD, HPA axis abnormalities are likely predicated on strong negative feedback inhibition of cortisol, itself likely due to an increased sensitivity of glucocorticoid receptors.^[85] PTSD has been hypothesized to be a

maladaptive learning pathway to fear response through a hypersensitive, hyperreactive, and hyperresponsive HPA axis.^[86]

Low cortisol levels may predispose individuals to PTSD: Following war trauma, Swedish soldiers serving in Bosnia and Herzegovina with low pre-service salivary cortisol levels had a higher risk of reacting with PTSD symptoms, following war trauma, than soldiers with normal pre-service levels.^[87] Because cortisol is normally important in restoring homeostasis after the stress response, it is thought that trauma survivors with low cortisol experience a poorly contained—that is, longer and more distressing—response, setting the stage for PTSD.

It is thought that the locus coeruleus-noradrenergic system mediates the over-consolidation of fear memory. High levels of cortisol reduce noradrenergic activity, and because people with PTSD tend to have reduced levels of cortisol, it has been proposed that individuals with PTSD cannot regulate the increased noradrenergic response to traumatic stress.^[88] Intrusive memories and conditioned fear responses are thought to be a result of the response to associated triggers. Neuropeptide Y has been reported to reduce the release of norepinephrine and has been demonstrated to have anxiolytic properties in animal models. Studies have shown people with PTSD demonstrate reduced levels of NPY, possibly indicating their increased anxiety levels.^[37]

Other studies indicate that people that suffer from PTSD have chronically low levels of serotonin, which contributes to the commonly associated behavioral symptoms such as anxiety, ruminations, irritability, aggression, suicidality, and impulsivity.^[89] Serotonin also contributes to the stabilization of glucocorticoid production.

Dopamine levels in a person with PTSD can help contribute to the symptoms associated. Low levels of dopamine can contribute to anhedonia, apathy, impaired attention, and motor deficits. Increased levels of dopamine can cause psychosis, agitation, and restlessness.^[89]

Multiple studies described elevated concentrations of the thyroid hormone triiodothyronine in PTSD.^[90] This kind of type 2 allostatic adaptation may contribute to increased sensitivity to catecholamines and other stress mediators.

Hyperresponsiveness in the norepinephrine system can also be caused by continued exposure to high stress. Overactivation of norepinephrine receptors in the prefrontal cortex can be connected to the flashbacks and nightmares frequently experienced by those with PTSD. A decrease in other norepinephrine functions (awareness of the current environment) prevents the memory mechanisms in the brain from processing that the experience, and emotions the person is experiencing during a flashback are not associated with the current environment.^[89]

There is considerable controversy within the medical community regarding the neurobiology of PTSD. A 2012 review showed no clear relationship between cortisol levels and PTSD. The majority of reports indicate people with PTSD have elevated levels of corticotropin-releasing hormone, lower basal cortisol levels, and enhanced negative feedback suppression of the HPA axis by dexamethasone.^{[37][91]}

Neuroanatomy

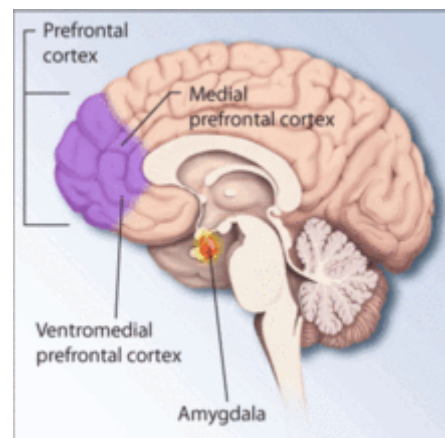
The three brain areas with changed function are the prefrontal cortex, amygdala, and hippocampus. Much of this research stems from PTSD victims from the Vietnam War.^{[93][94]}

PTSD patients have decreased brain activity in the dorsal and rostral anterior cingulate cortices and the ventromedial prefrontal cortex, areas linked to the experience and regulation of emotion.^[95]

The amygdala is strongly involved in forming emotional memories, especially fear-related memories. During high stress, the hippocampus, which is associated with placing memories in the correct context of space and time and memory recall, is suppressed. According to one theory this suppression may be the cause of the flashbacks that can affect people with PTSD. When someone with PTSD undergoes stimuli similar to the traumatic event, the body perceives the event as occurring again because the memory was never properly recorded in the person's memory.^{[37][96]}

The amygdalocentric model of PTSD proposes that the amygdala is very much aroused and insufficiently controlled by the medial prefrontal cortex and the hippocampus, in particular during extinction.^[97] This is consistent with an interpretation of PTSD as a syndrome of deficient extinction ability.^{[97][98]}

The basolateral nucleus (BLA) of the amygdala is responsible for the comparison and development of associations between unconditioned and conditioned responses to stimuli, which results in the fear conditioning present in PTSD. The BLA activates the central nucleus (CeA) of the amygdala, which elaborates the fear response, (including behavioral response to threat and elevated startle response). Descending inhibitory inputs from the medial prefrontal cortex (mPFC) regulate the transmission from the BLA to the CeA, which is hypothesized to play a role in the extinction of conditioned fear responses.^[37] While as a whole, amygdala hyperactivity is reported by meta analysis of functional neuroimaging in PTSD, there is a large degree of heterogeneity, more so than in social anxiety disorder or phobic disorder. Comparing dorsal(roughly the CeA) and ventral(roughly the BLA) clusters, hyperactivity is more robust in the ventral cluster, while hypoactivity is evident in the dorsal cluster. The distinction may explain the blunted emotions in PTSD(via desensitization in the CeA) as well as the fear related component.^[99]



Regions of the brain associated with stress and posttraumatic stress disorder^[92]

In a 2007 study Vietnam War combat veterans with PTSD showed a 20% reduction in the volume of their hippocampus compared with veterans having suffered no such symptoms.^[100] This finding was not replicated in chronic PTSD patients traumatized at an air show plane crash in 1988 (Ramstein, Germany).^[101]

Diagnosis

PTSD can be particularly difficult to diagnose, because numerous factors can lead to over-reporting (e.g., disability) and under-reporting (e.g., avoidance) symptoms, dysfunction and distress.

Screening and assessment

A number of screening instruments are used for screening adults for PTSD, such as the Clinician-Administered PTSD Scale for *DSM-5* (CAPS-5),^[102] Primary Care PTSD Screen for *DSM-5* (PC-PTSD-5),^{[103][104]} PTSD Checklist for *DSM-5* (PCL-5),^[105] and Dissociative Subtype of PTSD Scale (DSPS).^[106] The CAPS-5 is considered the gold-standard assessment recommended for use by the U.S. National Center for PTSD.^[102]

There are also several screening and assessment instruments for use with children and adolescents. These include the Child PTSD Symptom Scale (CPSS),^{[107][108]} Clinician-Administered PTSD Scale for *DSM-5* -Child/Adolescent version (CAPS-CA-5),^[109] Child Trauma Screening Questionnaire,^{[110][111]} and UCLA Posttraumatic Stress Disorder Reaction Index for *DSM-IV*.^{[112][113]}

Diagnostic and statistical manual

PTSD was classified as an anxiety disorder in the *DSM-IV*, but has since been reclassified as a "trauma- and stressor-related disorder" in the *DSM-5*.^[1] The *DSM-5* diagnostic criteria for PTSD include four symptom clusters: re-experiencing, avoidance, negative alterations in cognition/mood, and alterations in arousal and reactivity.^{[1][3]}

International classification of diseases

The International Classification of Diseases and Related Health Problems 10 (ICD-10) classifies PTSD under "Reaction to severe stress, and adjustment disorders."^[114] The ICD-10 criteria for PTSD include re-experiencing, avoidance, and either increased reactivity or inability to recall certain details related to the event.^[114]

Differential diagnosis

A diagnosis of PTSD requires that the person has been exposed to an extreme stressor such as one that is life-threatening. Any stressor can result in a diagnosis of adjustment disorder and it is an appropriate diagnosis for a stressor and a symptom pattern that does not meet the criteria for PTSD, for example a partner being fired, or a spouse leaving. If any of the symptom pattern is present before the stressor, another diagnosis is required, such as brief psychotic disorder or major depressive disorder. Other differential diagnoses are schizophrenia or other disorders with psychotic features such as Psychotic disorders due to a general medical condition. Drug-induced psychotic disorders can be considered if substance abuse is involved.^[17]

The symptom pattern for acute stress disorder must occur and be resolved within four weeks of the trauma. If it lasts longer, and the symptom pattern fits that characteristic of PTSD, the diagnosis may be changed.^[17]

Obsessive compulsive disorder may be diagnosed for intrusive thoughts that are recurring but not related to a specific traumatic event.^[17]

Prevention

Modest benefits have been seen from early access to cognitive behavioral therapy. Critical incident stress management has been suggested as a means of preventing PTSD, but subsequent studies suggest the likelihood of its producing negative outcomes.^{[115][116]} A review "...did not find any evidence to support the use of an intervention offered to everyone", and that "...multiple session interventions may result in worse outcome than no intervention for some individuals."^[117] The World Health Organization recommends against the use of benzodiazepines and antidepressants in those having experienced trauma.^[118] Some evidence supports the use of hydrocortisone for prevention in adults, however there is limited or no evidence supporting propranolol, escitalopram, temazepam, or gabapentin.^[119]

Psychological debriefing

Trauma-exposed individuals often receive treatment called *psychological debriefing* in an effort to prevent PTSD, which consists of interviews that are meant to allow individuals to directly confront the event and share their feelings with the counselor and to help structure their memories of the event.^[120] However, several meta-analyses find that psychological debriefing is unhelpful and is potentially harmful.^{[120][121][122]} This is true for both single-session debriefing and multiple session interventions.^[117] As of 2017 The American Psychological Association assessed psychological debriefing as *No Research Support/Treatment is Potentially Harmful*.^[123]

Risk-targeted interventions

Risk-targeted interventions are those that attempt to mitigate specific formative information or events. It can target modeling normal behaviors, instruction on a task, or giving information on the event.^{[124][125]}

Management

Reviews of studies have found that combination therapy (psychological and pharmacotherapy) is no more effective than psychological therapy alone.^[10]

Psychotherapy

Many forms of psychotherapy have been found to be efficacious for trauma-related problems such as PTSD. Basic counseling practices common to many treatments for PTSD include education about the condition, and provision of safety and support.^{[18][126]}

The psychotherapy approaches with the strongest demonstrated efficacy include cognitive behavioral therapy, prolonged exposure therapy,^[127] cognitive therapy (CT), cognitive processing therapy, and eye movement desensitization and reprocessing (EMDR).^{[128][129][130]}

A meta-analytic comparison of EMDR and cognitive behavioral therapy found both protocols indistinguishable in terms of effectiveness in treating PTSD; however, "the contribution of the eye movement component in EMDR to treatment outcome" is unclear.^[131] A meta-analysis in children and adolescent also found that EMDR was as efficacious as cognitive behavioral therapy (CBT).^[132]

Furthermore, the availability of school-based therapy is particularly important for children with PTSD. Children with PTSD are far more likely to pursue treatment at school (because of its proximity and ease) than at a free clinic.^[133]

Cognitive behavioral therapy

CBT seeks to change the way a person feels and acts by changing the patterns of thinking or behavior, or both, responsible for negative emotions. CBT has been proven to be an effective treatment for PTSD and is currently considered the standard of care for PTSD by the United States Department of Defense.^{[134][135]} In CBT, individuals learn to identify thoughts that make them feel afraid or upset and replace them with less distressing thoughts. The goal is to understand how certain thoughts about events cause PTSD-related stress.^{[136][137]}

Recent research on contextually based third-generation behavior therapies suggests that they may produce results comparable to some of the better validated therapies.^[138] Many of these therapy methods have a significant element of exposure^[139] and have demonstrated success in treating the primary problems of PTSD and co-occurring depressive symptoms.^[140]

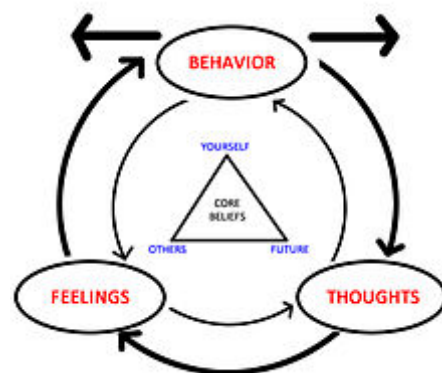
Exposure therapy is a type of cognitive behavioral therapy^[141] that involves assisting trauma survivors to re-experience distressing trauma-related memories and reminders in order to facilitate habituation and successful emotional processing of the trauma memory. Most exposure therapy programs include both imaginal confrontation with the traumatic memories and real-life exposure to trauma reminders; this therapy modality is well supported by clinical evidence. The success of exposure-based therapies has raised the question of whether exposure is a necessary ingredient in the treatment of PTSD.^[142] Some organizations have endorsed the need for exposure.^{[143][144]} The U.S. Department of Veterans Affairs has been actively training mental health treatment staff in prolonged exposure therapy^[145] and Cognitive Processing Therapy^[146] in an effort to better treat U.S. veterans with PTSD.

Eye movement desensitization and reprocessing

Eye movement desensitization and reprocessing (EMDR) is a form of psychotherapy developed and studied by Francine Shapiro.^[147] She had noticed that, when she was thinking about disturbing memories herself, her eyes were moving rapidly. When she brought her eye movements under control while thinking, the thoughts were less



An assistance dog trained to help veterans with PTSD



The diagram depicts how emotions, thoughts, and behaviors all influence each other. The triangle in the middle represents CBT's tenet that all humans' core beliefs can be summed up in three categories: self, others, future.

distressing.^[147]

In 2002, Shapiro and Maxfield published a theory of why this might work, called adaptive information processing.^[148] This theory proposes that eye movement can be used to facilitate emotional processing of memories, changing the person's memory to attend to more adaptive information.^[149] The therapist initiates voluntary rapid eye movements while the person focuses on memories, feelings or thoughts about a particular trauma.^{[8][150]} The therapists uses hand movements to get the person to move their eyes backward and forward, but hand-tapping or tones can also be used.^[8] EMDR closely resembles cognitive behavior therapy as it combines exposure (re-visiting the traumatic event), working on cognitive processes and relaxation/self-monitoring.^[8] However, exposure by way of being asked to think about the experience rather than talk about it has been highlighted as one of the more important distinguishing elements of EMDR.^[151]

There have been multiple small controlled trials of four to eight weeks of EMDR in adults^[152] as well as children and adolescents.^[150] EMDR reduced PTSD symptoms enough in the short term that one in two adults no longer met the criteria for PTSD, but the number of people involved in these trials was small.^[152] There was not enough evidence to know whether or not EMDR could eliminate PTSD in adults.^[152] In children and adolescents, a recent meta-analysis of randomized controlled trials using MetaNSUE to avoid biases related to missing information found that EMDR was at least as efficacious as CBT, and superior to waitlist or placebo.^[132] There was some evidence that EMDR might prevent depression.^[152] There were no studies comparing EMDR to other psychological treatments or to medication.^[152] Adverse effects were largely unstudied.^[152] The benefits were greater for women with a history of sexual assault compared with people who had experienced other types of traumatizing events (such as accidents, physical assaults and war). There is a small amount of evidence that EMDR may improve re-experiencing symptoms in children and adolescents, but EMDR has not been shown to improve other PTSD symptoms, anxiety, or depression.^[150]

The eye movement component of the therapy may not be critical for benefit.^{[8][149]} As there has been no major, high quality randomized trial of EMDR with eye movements versus EMDR without eye movements, the controversy over effectiveness is likely to continue.^[151] Authors of a meta-analysis published in 2013 stated, "We found that people treated with eye movement therapy had greater improvement in their symptoms of post-traumatic stress disorder than people given therapy without eye movements....Secondly we found that that in laboratory studies the evidence concludes that thinking of upsetting memories and simultaneously doing a task that facilitates eye movements reduces the vividness and distress associated with the upsetting memories."^[129]

Interpersonal psychotherapy

Other approaches, in particular involving social supports,^{[153][154]} may also be important. An open trial of interpersonal psychotherapy^[155] reported high rates of remission from PTSD symptoms without using exposure.^[156] A current, NIMH-funded trial in New York City is now (and into 2013) comparing interpersonal psychotherapy, prolonged exposure therapy, and relaxation therapy.^{[157][158][159]}

Medication

While many medications do not have enough evidence to support their use, three (fluoxetine, paroxetine, and venlafaxine) have been shown to have a small benefit over placebo.^[12] With many medications, residual PTSD symptoms following treatment is the rule rather than the exception.^[160]

Antidepressants

Selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) may have some benefit for PTSD symptoms.^{[12][161]} Tricyclic antidepressants are equally effective but are less well tolerated.^[162] Evidence provides support for a small or modest improvement with sertraline, fluoxetine, paroxetine, and

venlafaxine.^{[12][163]} Thus, these four medications are considered to be first-line medications for PTSD.^{[161][164]}

Benzodiazepines

Benzodiazepines are not recommended for the treatment of PTSD due to a lack of evidence of benefit and risk of worsening PTSD symptoms.^[165] Some authors believe that the use of benzodiazepines is contraindicated for acute stress, as this group of drugs promotes dissociation and ulterior revivals.^[166] Nevertheless, some use benzodiazepines with caution for short-term anxiety and insomnia.^{[167][168][169]} While benzodiazepines can alleviate acute anxiety, there is no consistent evidence that they can stop the development of PTSD and may actually increase the risk of developing PTSD 2–5 times.^[11] Additionally, benzodiazepines may reduce the effectiveness of psychotherapeutic interventions, and there is some evidence that benzodiazepines may actually contribute to the development and chronification of PTSD. For those who already have PTSD, benzodiazepines may worsen and prolong the course of illness, by worsening psychotherapy outcomes, and causing or exacerbating aggression, depression (including suicidality), and substance use.^[11] Drawbacks include the risk of developing a benzodiazepine dependence, tolerance (i.e., short-term benefits wearing off with time), and withdrawal syndrome; additionally, individuals with PTSD (even those without a history of alcohol or drug misuse) are at an increased risk of abusing benzodiazepines.^{[164][170]} Due to a number of other treatments with greater efficacy for PTSD and less risks (e.g., prolonged exposure, cognitive processing therapy, eye movement desensitization and reprocessing, cognitive restructuring therapy, trauma-focused cognitive behavioral therapy, brief eclectic psychotherapy, narrative therapy, stress inoculation training, serotonergic antidepressants, adrenergic inhibitors, antipsychotics, and even anticonvulsants), benzodiazepines should be considered relatively contraindicated until all other treatment options are exhausted.^{[9][171]} For those who argue that benzodiazepines should be used sooner in the most severe cases, the adverse risk of disinhibition (associated with suicidality, aggression and crimes) and clinical risks of delaying or inhibiting definitive efficacious treatments, make other alternative treatments preferable (e.g., inpatient, residential, partial hospitalization, intensive outpatient, dialectic behavior therapy; and other fast-acting sedating medications such as trazodone, mirtazapine, amitriptyline, doxepin, prazosin, propranolol, guanfacine, clonidine, quetiapine, olanzapine, valproate, gabapentin).^{[4][171][172]}

Glucocorticoids

Glucocorticoids may be useful for short-term therapy to protect against neurodegeneration caused by the extended stress response that characterizes PTSD, but long-term use may actually promote neurodegeneration.^[173]

Cannabinoids

Evidence as of 2017 is insufficient to determine if medical cannabis useful for PTSD.^[174] Despite the uncertain evidence, use of cannabis or derived products is widespread among U.S. veterans with PTSD.^[175]

The cannabinoid nabilone is sometimes used off-label for nightmares in PTSD. Although some short-term benefit was shown, adverse effects are common and it has not been adequately studied to determine efficacy.^[176] Additionally, there are other treatments with stronger efficacy and less risks (e.g., psychotherapy, serotonergic antidepressants, adrenergic inhibitors). The use of medical marijuana for PTSD is controversial, with only a handful of states permitting its use for that purpose.^[177]

Other

Exercise, sport and physical activity

Physical activity can influence people's psychological^[178] and physical health.^[179] The U.S. National Center for PTSD recommends moderate exercise as a way to distract from disturbing emotions, build self-esteem and increase feelings of being in control again. They recommend a discussion with a doctor before starting an exercise program.^[180]

Play therapy for children

Play is thought to help children link their inner thoughts with their outer world, connecting real experiences with abstract thought.^[181] Repetitive play can also be one way a child relives traumatic events, and that can be a symptom of trauma in a child or young person.^[182] Although it is commonly used, there have not been enough studies comparing outcomes in groups of children receiving and not receiving play therapy, so the effects of play therapy are not yet understood.^{[8][181]}

Military programs

Many veterans of the wars in Iraq and Afghanistan have faced significant physical, emotional, and relational disruptions. In response, the United States Marine Corps has instituted programs to assist them in re-adjusting to civilian life, especially in their relationships with spouses and loved ones, to help them communicate better and understand what the other has gone through.^[183] Walter Reed Army Institute of Research (WRAIR) developed the Battlemind program to assist service members avoid or ameliorate PTSD and related problems. Wounded Warrior Project partnered with the US Department of Veterans Affairs to create Warrior Care Network, a national health system of PTSD treatment centers.^{[184][185]}

Epidemiology

There is debate over the rates of PTSD found in populations, but, despite changes in diagnosis and the criteria used to define PTSD between 1997 and 2013, epidemiological rates have not changed significantly.^{[187][188]} Most of the current reliable data regarding the epidemiology of PTSD is based on DSM-IV criteria, as the DSM-5 was not introduced until 2013.

The United Nations' World Health Organization publishes estimates of PTSD impact for each of its member states; the latest data available are for 2004. Considering only the 25 most populated countries ranked by overall age-standardized Disability-Adjusted Life Year (DALY) rate, the top half of the ranked list is dominated by Asian/Pacific countries, the US, and Egypt.^[189] Ranking the countries by the male-only or female-only rates produces much the same result, but with less meaningfulness, as the score range in the single-sex rankings is much-reduced (4 for women, 3 for men, as compared with 14 for the overall score range), suggesting that the differences between female and male rates, within each country, is what drives the distinctions between the countries.^{[190][191]}



Disability-adjusted life year rates for posttraumatic stress disorder per 100,000 inhabitants in 2004.^[186]

<div></div>	no data	<div></div>	51–52.5
<div></div>	< 43.5	<div></div>	52.5–54
<div></div>	43.5–45	<div></div>	54–55.5
<div></div>	45–46.5	<div></div>	55.5–57
<div></div>	46.5–48	<div></div>	57–58.5
<div></div>	48–49.5	<div></div>	> 58.5
<div></div>	49.5–51		

Age-standardized Disability-adjusted life year (DALY) rates for PTSD, per 100,000 inhabitants, in 25 most populous countries, ranked by overall rate (2004)

Region	Country	PTSD DALY rate, overall ^[189]	PTSD DALY rate, females ^[190]	PTSD DALY rate, males ^[191]
Asia / Pacific	Thailand	59	86	30
Asia / Pacific	Indonesia	58	86	30
Asia / Pacific	Philippines	58	86	30
Americas	USA	58	86	30
Asia / Pacific	Bangladesh	57	85	29
Africa	Egypt	56	83	30
Asia / Pacific	India	56	85	29
Asia / Pacific	Iran	56	83	30
Asia / Pacific	Pakistan	56	85	29
Asia / Pacific	<u>Japan</u>	55	80	31
Asia / Pacific	Myanmar	55	81	30
Europe	Turkey	55	81	30
Asia / Pacific	Vietnam	55	80	30
Europe	France	54	80	28
Europe	Germany	54	80	28
Europe	Italy	54	80	28
Asia / Pacific	Russian Federation	54	78	30
Europe	United Kingdom	54	80	28
Africa	Nigeria	53	76	29
Africa	Dem. Republ. of Congo	52	76	28
Africa	Ethiopia	52	76	28
Africa	South Africa	52	76	28
Asia / Pacific	China	51	76	28
Americas	Mexico	46	60	30
Americas	Brazil	45	60	30

United States

The National Comorbidity Survey Replication has estimated that the lifetime prevalence of PTSD among adult Americans is 6.8%, with women (9.7%) more than twice as likely as men^[89] (3.6%) to have PTSD at some point in their lives.^[41] More than 60% of men and more than 60% of women experience at least one traumatic event in their life. The most frequently reported traumatic events by men are rape, combat, and childhood neglect or physical abuse. Women most frequently report instances of rape, sexual molestation, physical attack, being threatened with a weapon and childhood physical abuse.^[89] 88% of men and 79% of women with lifetime PTSD have at least one comorbid psychiatric disorder. Major depressive disorder, 48% of men and 49% of women, and lifetime alcohol abuse or dependence, 51.9% of men and 27.9% of women, are the most common comorbid disorders.^[192]

The United States Department of Veterans Affairs estimates that 830,000 Vietnam War veterans suffered symptoms of PTSD.^[193] The *National Vietnam Veterans' Readjustment Study* (NVVRS) found 15.2% of male and 8.5% of female Vietnam veterans to suffer from current PTSD at the time of the study. Life-Time prevalence of PTSD was 30.9% for

males and 26.9% for females. In a reanalysis of the NVVRS data, along with analysis of the data from the Matsunaga Vietnam Veterans Project, Schnurr, Lunney, Sengupta, and Waelde found that, contrary to the initial analysis of the NVVRS data, a large majority of Vietnam veterans suffered from PTSD symptoms (but not the disorder itself). Four out of five reported recent symptoms when interviewed 20–25 years after Vietnam.^[194]

A 2011 study from [Georgia State University](#) and [San Diego State University](#) found that rates of PTSD diagnosis increased significantly when troops were stationed in combat zones, had tours of longer than a year, experienced combat, or were injured. Military personnel serving in combat zones were 12.1 percentage points more likely to receive a PTSD diagnosis than their active-duty counterparts in non-combat zones. Those serving more than 12 months in a combat zone were 14.3 percentage points more likely to be diagnosed with PTSD than those having served less than one year. Experiencing an enemy firefight was associated a 18.3 percentage point increase in the probability of PTSD, while being wounded or injured in combat was associated a 23.9 percentage point increase in the likelihood of a PTSD diagnosis. For the 2.16 million U.S. troops deployed in combat zones between 2001 and 2010, the total estimated two-year costs of treatment for combat-related PTSD are between \$1.54 billion and \$2.69 billion.^[195]

As of 2013, rates of PTSD have been estimated at up to 20% for veterans returning from Iraq and Afghanistan.^[196] As of 2013 13% of veterans returning from Iraq were [unemployed](#).^[197]

Veterans

United States

The United States provides a range of benefits for veterans that the [VA](#) has determined have PTSD, which developed during, or as a result of, their military service. These benefits may include tax-free cash payments,^[198] free or low-cost mental health treatment and other healthcare,^[199] vocational rehabilitation services,^[200] employment assistance,^[201] and independent living support.^{[202][203]}

United Kingdom

In the UK, there are various charities and service organisations dedicated to aiding veterans in readjusting to civilian life. [The Royal British Legion](#) and the more recently established [Help for Heroes](#) are two of Britain's more high-profile veterans' organisations which have actively advocated for veterans over the years. There has been some controversy that the [NHS](#) has not done enough in tackling mental health issues and is instead "dumping" veterans on charities such as [Combat Stress](#).^{[204][205]}

Canada

[Veterans Affairs Canada](#) offers a new program that includes rehabilitation, financial benefits, job placement, health benefits program, disability awards, [peer support](#)^{[206][207][208]} and family support.^[209]

History

The 1952 edition of the DSM-I includes a diagnosis of "gross stress reaction", which has similarities to the modern definition and understanding of PTSD.^[210] Gross stress reaction is defined as a "normal personality [utilizing] established patterns of reaction to deal with overwhelming fear" as a response to "conditions of great stress".^[211] The diagnosis includes language which relates the condition to combat as well as to "civilian catastrophe".^[211]



Vietnam Veterans Memorial, Washington, D.C.

Early in 1978, the term was used in a working group finding presented to the Committee of Reactive Disorders.^[212] The condition was added to the DSM-III, which was being developed in the 1980s, as posttraumatic stress disorder.^{[210][212]} In the DSM-IV, the spelling "posttraumatic stress disorder" is used, while in the ICD-10, the spelling is "post-traumatic stress disorder".^[213]

The addition of the term to the DSM-III was greatly influenced by the experiences and conditions of U.S. military veterans of the Vietnam War.^[214] Due to its association with the war in Vietnam, PTSD has become synonymous with many historical war-time diagnoses such as railway spine, stress syndrome, nostalgia, soldier's heart, shell shock, battle fatigue, combat stress reaction, or traumatic war neurosis.^{[215][216]} Some of these terms date back to the 19th century, which is indicative of the universal nature of the condition. In a similar vein, psychiatrist Jonathan Shay has proposed that Lady Percy's soliloquy in the William Shakespeare play Henry IV, Part 1 (act 2, scene 3, lines 40–62^[217]), written around 1597, represents an unusually accurate description of the symptom constellation of PTSD.^[218]

The correlations between combat and PTSD are undeniable; according to Stéphane Audoin-Rouzeau and Annette Becker, "One-tenth of mobilized American men were hospitalized for mental disturbances between 1942 and 1945, and, after thirty-five days of uninterrupted combat, 98% of them manifested psychiatric disturbances in varying degrees."^[219] In fact, much of the available published research regarding PTSD is based on studies done on veterans of the war in Vietnam. A study based on personal letters from soldiers of the 18th-century Prussian Army concludes that combatants may have had PTSD.^[220]



Statue, *Three Servicemen*, Vietnam Veterans Memorial

The researchers from the Grady Trauma Project highlight the tendency people have to focus on the combat side of PTSD: "less public awareness has focused on civilian PTSD, which results from trauma exposure that is not combat related... " and "much of the research on civilian PTSD has focused on the sequelae of a single, disastrous event, such as the Oklahoma City bombing, September 11th attacks, and Hurricane Katrina".^[221] Disparity in the focus of PTSD research affects the already popular perception of the exclusive interconnectedness of combat and PTSD. This is misleading when it comes to understanding the implications and extent of PTSD as a neurological disorder. Dating back to the definition of Gross stress reaction in the DSM-I, civilian experience of catastrophic or high stress events is included as a cause of PTSD in medical literature. The 2014 National Comorbidity Survey reports that "the traumas most commonly associated with PTSD are combat exposure and witnessing among men and rape and sexual molestation among women."^[222] Because of the initial overt focus on PTSD as a combat related disorder when it was first fleshed out in the years following the war in Vietnam, in 1975 Ann Wolbert Burgess and Lynda Lytle Holmstrom defined Rape trauma syndrome, RTS, in order to draw attention to the striking similarities between the experiences of soldiers returning from war and of rape victims.^[223] This paved the way for a more comprehensive understanding of causes of PTSD.

The DSM-IV classified PTSD under anxiety disorders, but the DSM-5 created a new category called "Trauma- and Stressor-Related Disorders," in which PTSD is now classified.^[1]

Terminology

The Diagnostic and Statistical Manual of Mental Disorders does not hyphenate 'post' and 'traumatic', thus, the DSM-5 lists the disorder as *posttraumatic stress disorder*. However, many scientific journal articles and other scholarly publications do hyphenate the name of the disorder, *viz.*, post-traumatic stress disorder.^[224] Dictionaries also differ with regard to the preferred spelling of the disorder with the *Collins English Dictionary - Complete and Unabridged* using the hyphenated spelling, and the *American Heritage Dictionary of the English Language, Fifth Edition* and the *Random House Kernerman Webster's College Dictionary* giving the non-hyphenated spelling.^[225]

Research

Most knowledge regarding PTSD comes from studies in high-income countries.^[226]

To recapitulate some of the neurological and neurobehavioral symptoms experienced by the veteran population of recent conflicts in Iraq and Afghanistan, researchers at the Roskamp Institute and the James A Haley Veteran's Hospital (Tampa) have developed an animal model to study the consequences of mild traumatic brain injury (mTBI) and PTSD.^[227] In the laboratory, the researchers exposed mice to a repeated session of unpredictable stressor (i.e. predator odor while restrained), and physical trauma in the form of inescapable foot-shock, and this was also combined with a mTBI. In this study, PTSD animals demonstrated recall of traumatic memories, anxiety, and an impaired social behavior, while animals subject to both mTBI and PTSD had a pattern of disinhibitory-like behavior. mTBI abrogated both contextual fear and impairments in social behavior seen in PTSD animals. In comparison with other animal studies,^{[227][228]} examination of neuroendocrine and neuroimmune responses in plasma revealed a trend toward increase in corticosterone in PTSD and combination groups.

Psychotherapy adjuncts

MDMA was used for psychedelic therapy for a variety of indications before its criminalization in the U.S. in 1985. In response to its criminalization, the Multidisciplinary Association for Psychedelic Studies was founded as a nonprofit drug-development organization to develop MDMA into a legal prescription drug for use as an adjunct in psychotherapy.^[229] The drug is hypothesized to facilitate psychotherapy by reducing fear, thereby allowing people to reprocess and accept their traumatic memories without becoming emotionally overwhelmed. In this treatment, people participate in an extended psychotherapy session during the acute activity of the drug, and then spend the night at the treatment facility. In the sessions with the drug, therapists are not directive and support the patients in exploring their inner experiences. People participate in standard psychotherapy sessions before the drug-assisted sessions, as well as after the drug-assisted psychotherapy to help them integrate their experiences with the drug.^[230] The phase 2 clinical trials of the MDMA-Assisted Psychotherapy, was publicized at the end of November 2016.^[231] Preliminary results suggest MDMA-assisted psychotherapy might be effective.^[232] MAPS is currently waiting for the FDA approval of the phase 3 as of early 2017.^[233]

Research is also investigating using D-cycloserine, hydrocortisone, and propranolol as add on therapy to more conventional exposure therapy.^[234]


Lawsuits

PTSD may lead to lawsuits.^[235]



Notes




- Acceptable variants of this term exist; see the *Terminology* section in this article.




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





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



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External links

-  Psychiatry portal
- Posttraumatic stress disorder (https://curlie.org/Health/Mental_Health/Disorders/Anxiety/Post-traumatic_Stress) at Curlie (based on DMOZ)
- Resources for the public (<http://www.ptsd.va.gov/public/index.asp>) from VA National PTSD Center (<http://www.ptsd.va.gov/index.asp>)
- Resources for professionals (<http://www.ptsd.va.gov/professional/index.asp>) from VA National PTSD Center (<http://www.ptsd.va.gov/index.asp>)
- Post Traumatic Stress Disorder Information Resource (<http://www.som.uq.edu.au/ptsd>) from The University of Queensland School of Medicine (<http://www.uq.edu.au/>)
- APA practice parameters for assessment and treatment for PTSD (Updated 2017) (<http://www.apa.org/ptsd-guideline/>)

Classification	ICD-10: V · T · D F43.1 (http://apps.who.int/classification/icd10/browse/2016/en#/F43.1) · ICD-9-CM: 309.81 (http://www.icd9data.com/getICD9Code.aspx?icd9=309.81) · MeSH: D013313 (https://www.nlm.nih.gov/cgi/mesh/2015/MB_cgi?field=uid&term=D013313) · DiseasesDB: 33846 (http://www.diseasesdatabase.com/ddb33846.htm)
External resources	MedlinePlus: 000925 (https://www.nlm.nih.gov/medlineplus/ency/article/000925.htm) ·

eMedicine:

med/1900 (<http://www.emedicine.com/med/topic1900.htm>)

• Patient UK:

Posttraumatic stress disorder (<http://patient.info/doctor/post-traumatic-stress-disorder-pro>)

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